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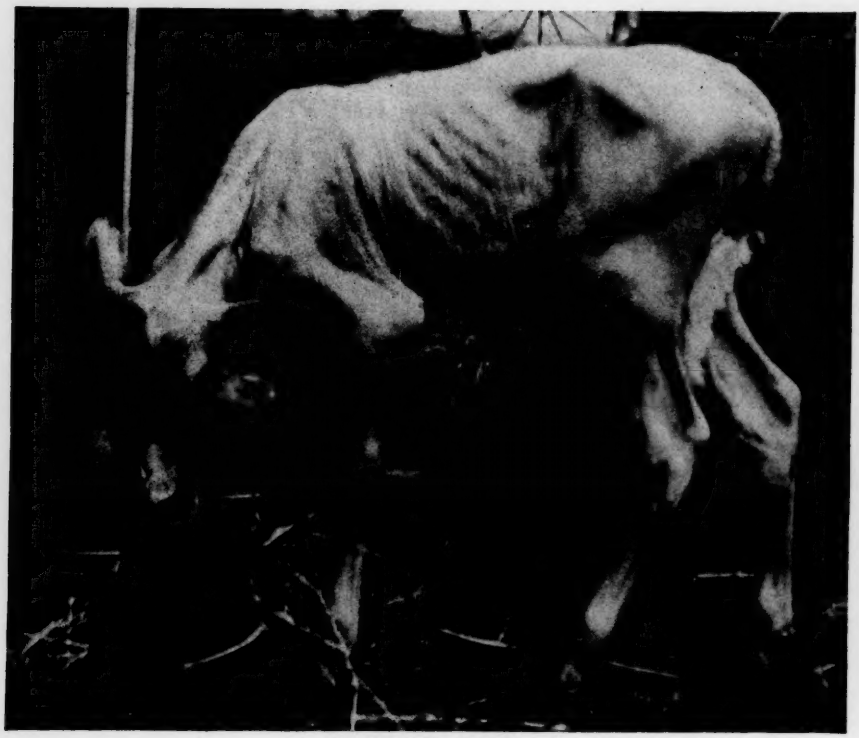

HAIRLESS CALF

(SEE PAGE 175)

Ataxic Pigeons

Haploid Sorghum

Antigens of Rabbits and Men

VOLUME 34 JUNE, 1943 NUMBER 6

The American Genetic Association

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The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore, Md., and 1108 Sixteenth St., N. W., Washington, D. C. Address all communications regarding editorial matters to Room 205, 1108 Sixteenth St., N. W., Washington, D. C. Communications regarding the business management of The JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to 1108 Sixteenth Street, N. W., Washington, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103, Act of October 3, 1917, authorized December 20, 1918.

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$3.50 a Year. 35c a Copy. Prices of back numbers on application.

The

Journal of Heredity

Vol. XXXIV **JUNE, 1943** **No. 6**

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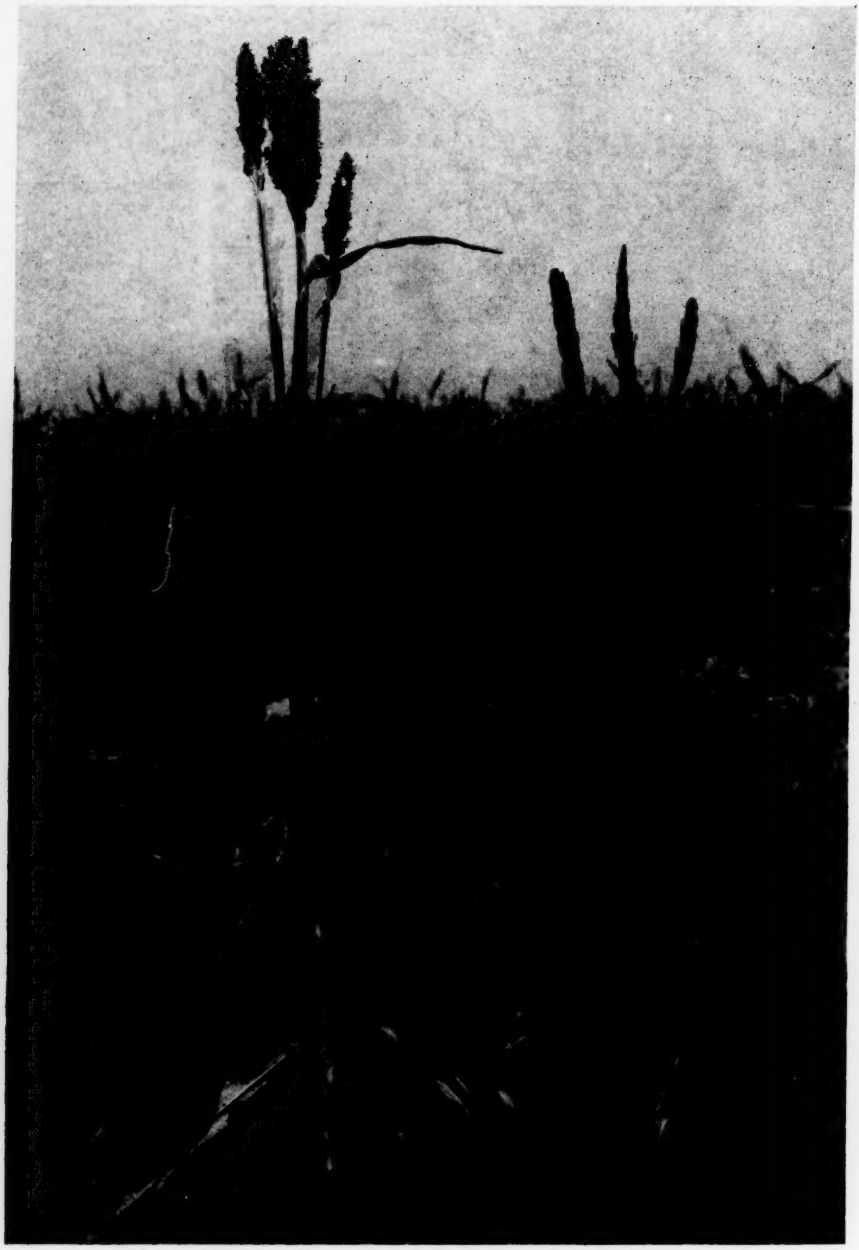


NORMAL SORGHUM PLANT COMPARED WITH HAPLOID

Figure 1

A haploid plant of Blackhull kafir having only a single set of chromosomes, compared with a normal diploid (paired-chromosome set) plant at the left. The slender growth habit, and the seedless flower-heads are characteristic of the haploids.

HAPLOID PLANTS IN SORGHUM

META SUCHE BROWN
Division of Agronomy, Texas Agricultural Experiment Station

FOR the past ten or twelve years abnormal plants have been observed each season in field plantings of grain and forage sorghums grown at Chillicothe, Texas. These plants are characterized by narrow and numerous side branches with small slender heads and almost complete sterility. It was surmised that these plants might be haploids. Two such plants were analyzed cytologically. One plant, which appeared among the F_3 progeny of Blackhull kafir × Feterita in 1940, was carried through the winter in the greenhouse and grown again in the field in 1941; the other plant was found in a genetic stock in 1941. Pollen mother cell preparations showing ten univalent chromosomes at metaphase verified the haploid condition of both plants.

Description and Fertility of Haploid Plants

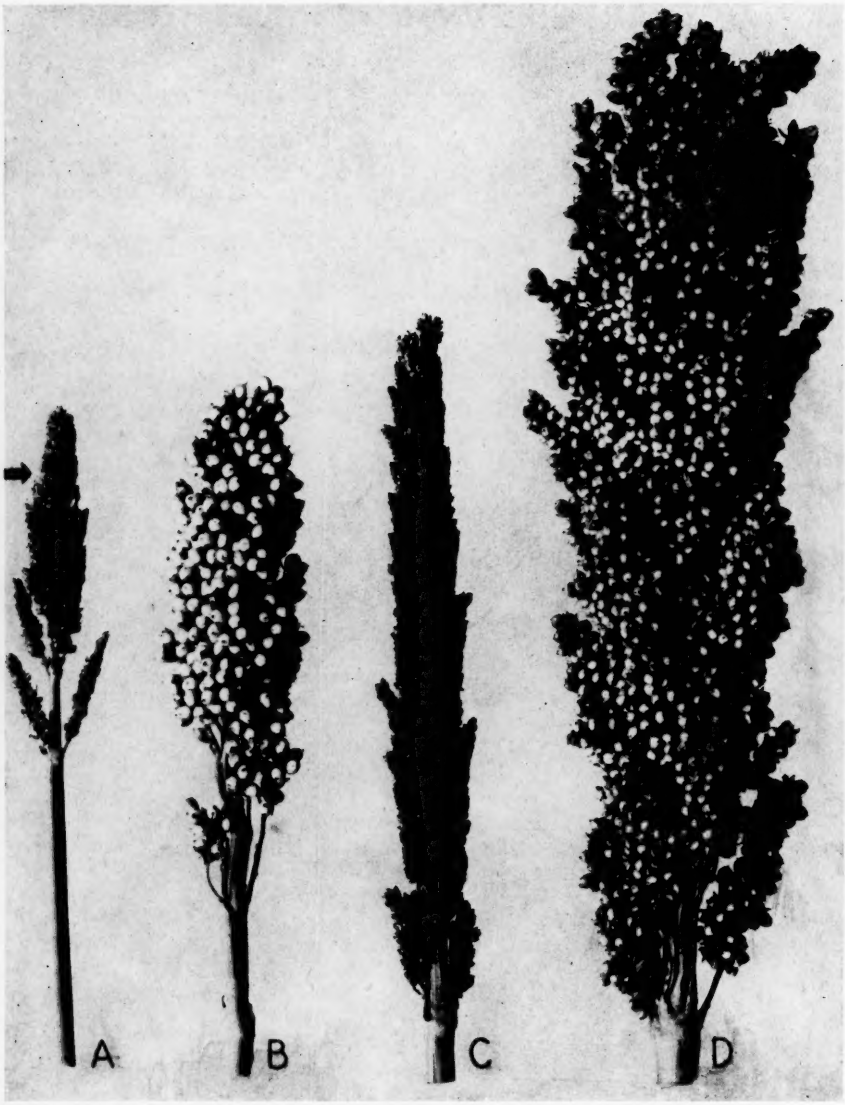
Haploid sorghum plants are shorter and less vigorous than their diploid siblings. They can be distinguished from normal plants by their relatively slender stalks, narrow leaves, small highly sterile panicles, and particularly by their small glumes. Spikelet structures within the outer glumes are reduced in size proportionately (Figures 1, 2, and 3). Excessive side-branching of haploids (Figure 3) is probably caused by incomplete fruiting in a manner similar to the excessive side-branching of diploid plants which have poor seed sets as a result of drought, midge damage, or male sterility. The stomatal guard cells are measurably smaller than in diploid plants (Table I). Most of the pollen grains produced by the haploid plants are empty and a large proportion are collapsed. Measurements of empty grains not collapsed show these to be smaller than diploid grains. A few filled grains are found among haploid pollen, and heads show an occasional seed.

Of the two seeds produced on five heads of one haploid, one germinated and produced a normal appearing plant. Cytological examination of pollen mother cells of this plant showed ten bivalents at diakinesis and metaphase, the normal diploid condition.

Cytological Behavior

Pollen mother cells produced by haploid sorghum are small and relatively few in number. Mid-prophase nuclei were generally unfavorable for study, but in two well stained nuclei the chromosomes were almost completely synapsed. The chromosomes were distinctly double. They were thick and deeply stained in the median regions adjoining the clear centromeres but threadlike and chromomeric in structure in the distal regions. In many regions the chromomeres were perfectly matched. At the unsynapsed end of one unequal pair only the shorter chromosome ended with a distinct chromomere.

At a stage corresponding to diakinesis ten univalents are present. The chromosomes are compact and rounded with no indication of the double nature which is evident in the chromosomes of the diploid at this stage. At metaphase most of the univalents are found on or near the equatorial plate. At a slightly later stage they are scattered on the spindle, and at anaphase are distributed at random to the two poles. Counts of anaphase separation range from 5 - 5 to 0 - 10, with 4 - 6 and 3 - 7 very common. Occasionally one or more bivalents are found at metaphase. Among approximately 150 metaphase nuclei, one case of three bivalents and four univalents, two cases of two bivalents and six univalents, and 13 cases of one bivalent and eight univalents were found. At anaphase six nuclei showing a bridge were found among 76 nuclei. One of these included a distinct fragment. One nucleus was found with 2 - 3 bridges and one with five chromatic strands stretched between the two masses



COMPARISON OF SEED HEADS FROM HAPLOID AND DIPLOID SORGHUM

Figure 2

A—haploid branch head showing one seed—indicated by arrow; *B*—diploid branch head (kafir); *C*—haploid main head; *D*—diploid main head (kafir). One of the occasional seeds in haploid heads has germinated, and produced a normal-appearing diploid plant.

OLD HAPLOID

Figure 3

Two year old haploid showing numerous side branches and small heads.

of chromatin at the poles. No evidence of univalents dividing at the first division was found.

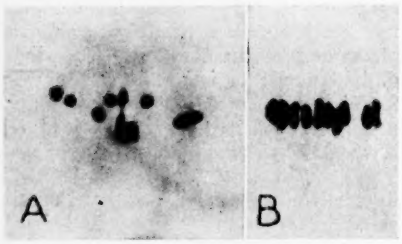
A photograph of haploid sorghum showing one bivalent and eight univalents at metaphase is shown in Figure 4*A*. Figure 4*B* shows normal metaphase with ten bivalents in a diploid.

The second meiotic division in haploid sorghum proceeds normally. The chromosomes line up on the equator, divide and pass to the poles. One case was found of a bridge persisting from first anaphase stretched across the cytoplasmic wall between the two equatorial plates of second metaphase. Occasional diploid nuclei of uncertain origin were found. One showed ten bivalents on the metaphase plate, and two showed ten bivalents disjoining at early anaphase. Three nuclei were found with 18 to 20 unpaired chromosomes scattered on the spindle.

Discussion

Cytological analysis of the abnormal sorghum plants has verified the supposition based on gross morphological observation that haploid plants occur in sorghum species as well as in other grasses. Müntzing[9] has reported one or more haploids among twin seedlings in the following species: *Triticum vulgare, Hordeum vulgare, Phleum pratense, Dactylis glomerata,* and *Poa pratensis.* The rice haploid reported by Ramiah *et al*[12] appeared likewise among twin seedlings. Haploids have been reported in some of these and in other genera following various forms of treatment. Rye haploids were found by Müntzing[8] following low temperature and by Nordenskiöld[11] following heat treatment. Katayama[6] found haploids of *Triticum monococcum* in field plantings and following X-ray treatments. Gaines and Aase[2] described haploids of *T. compactum,* and Nakajima[10] of *T. turgidum,* following intergeneric crosses. The haploids of *T. vulgare* described by Yamasaki[13] were found among strains of varietal crosses. In sorghum, probable haploids have been found by J. C. Stephens (unpublished) in many pure varieties, including Dwarf Yellow milo, Texas Blackhull kafir, Kansas Orange sorgo, and in numerous hybrid and genetic strains.

Twin-stalked seedlings with a single primary root and with both culms extending from within a single coleoptile are rather common in sorghum, 16 having been found at Chillicothe, Texas, in 1940. One of these was in the five-chromosome sorghum species *Sorghum versicolor,* five were in standard sor-



REDUCTION DIVISIONS

Figure 4

A—Metaphase of haploid sorghum showing one bivalent and eight univalents. Comparable diploid metaphase is shown in *B*, with complete and regular pairing of the homologous chromosomes.

ghum varieties, and the rest were in genetic and breeding stocks. Both stalks of most twins appear to be diploids but occasionally one stalk appears normal and the other has all of the morphological characteristics of a haploid. One twin, found in 1939 in a row segregating for a chlorophyl deficiency, had one green stalk, probably diploid and one chlorophyl deficient stalk which appeared haploid. Frequently the haploid-appearing stalk of twins fails to survive to the heading stage.

The meiotic chromosome behavior of sorghum haploids parallels that in other grass haploids. Univalents may all line up on the equatorial plate at metaphase, but at anaphase they are distributed at random as in other forms. No evidence was found of univalents dividing at first metaphase, as reported occasionally in wheat[1,2,11] and rarely in rye[11] and rice.[12]

The formation of bivalents in sorghum haploids is in line with the suggestion of Karper[4] and Karper and Chisholm,[5] following the discovery of a five-chromosome species of sorghum by Longley,[7] that five is the basic chromosome number in the sorghums. Huskins and Smith[3] interpreted the occurrence of duplicate or polymeric factors and the presence of quadrivalents in diploid forms as evidence of polyploidy in the sorghum genus.

The writer is indebted to J. C. Stephens of Texas Substation No. 12 at Chillicothe, Texas, for making the above material available for study.

Summary

The occurrence of haploids in sorghum is reported. Cytological study of meiosis shows the presence of ten univalents at late prophase. At metaphase bivalents are found in approximately ten per cent of the nuclei. Rarely diploid nuclei are found, which give rise to normal haploid pollen. The production of occasional diploid plants from haploids indicates that diploid nuclei with the full haploid complement of chromosomes are formed also during ovule development.

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TABLE I. Length of Stomatal Guard Cells.

Microns	16.7	20.0	23.3	26.7	30.0	33.3	36.7	40.0	43.3	46.7
Haploid No. 1 from F_3 of Blackhull kafir x feterita	3	49	92	15	1					
Haploid No. 2			13	33	86	80	8			
Diploid F_4 of haploid No. 1					5	24	62	53	15	1
Spur feterita					11	46	61	31	11	
Blackhull kafir				5	13	28	18	13	4	1

"SCRAGGLY" PLUMAGE AND ATAXIA

Two Inherited Characters in the Pigeon

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SINCE 1914 two mutational types which appeared in this Department's colony of pigeons have been bred and the normal and mutant offspring have been utilized for both genetic and physiological studies. In one of these types the defective plumage (scraggly) prevents flight and is little able to conserve heat; in the other type (ataxia) the underdevelopment of neural areas and tracts in brain and cord prevents a complete coordination of bodily movements and provides a basis for further functional changes or adjustments. The results of physiological studies on these types will be published elsewhere. An analysis of the inheritance of these two traits is presented here.

All matings have been kept in separate cages in order that parentage may be known definitely. However, the male parent for one mating in the fourth generation of the ataxia line is uncertain.

Scraggly

The second scraggly specimen that was reared to maturity and the first one bred was a male, *A126*, hatched in 1915. The sire of this bird was a checkered blue common pigeon with some white flight feathers, the dam blue with black bars. The latter pigeon, *545*, together with a scraggly brother (hatched, 1914) and five other normal male and female sibs, were from eggs produced by a mother subjected daily to alcohol fumes. Moreover, *545* was in turn treated with alcohol, and later with ether; her scraggly son, *A126*, was from such an "alcoholized" egg, and a scraggly daughter was from an "etherized egg; six other "treated" eggs produced normal offspring. For a time it was thought that such treatments or other adverse conditions were responsible for the scraggly character (e.g., Hoshino,[2] p. 113), but it soon became apparent that the trait had an hereditary basis. Prior to 1914 at least one or two cases of scraggly plumage had been observed by the senior author, but these birds had died while immature.

When scraggly ♂ *A126* became mature the original ataxic female *151* was already in reproduction and she was given as mate in order that the inheritance of the two characters could thus be studied together. These two birds were probably related, but the exact relationship is unknown. This mating (♂ *126* scraggly × ♀ *151* ataxic) produced 35 young, all normal in plumage and of both sexes. In the F_2 generation the scraggly trait reappeared frequently, as well as in a back cross of *A126* with a daughter. In Table I the breeding records involving the trait are summarized up to date. It is clear that the requirements for a simple recessive Mendelian factor are fulfilled, and there is no evidence of sex-linkage.

It is our experience that at hatching the scraggly squabs cannot be distinguished from normal. After about three days, however, close inspection shows that certain areas of the skin begin to look scaly or flaky; soon thereafter the entire skin becomes calloused, and crusty formations and scaling are wide-spread. The skin seems less flexible and the crop does not bulge out with food as it does in normal squabs (see Figure 5*A*) This condition is apparently the homologue of ichthyosis in man. No unusual parasites have been observed on the scraggly birds, and it is clearly not a contagious disease.

The plumage is slow in appearing. Some feathers may fail to pierce the skin but grow for a while beneath it. The squabs look miserable and require nor-

mally feathered foster parents to keep them warm. The ichthyosis persists throughout life, with some fluctuation. The feathers finally attain normal size but they are somewhat hairy in appearance and so defective that they function only partly as feathers (see Figure 5*B*, *D*). Flight is impossible though the wing movements are normal.

Examination of the feathers under a dissecting microscope reveals that initially the barbules are not absent but mainly they have failed to spread and interlock; they remain compressed against the sides and upper surface of the barb in the position assumed during early growth. When roughed up with a needle they fail to take the normal orientation; they are rather brittle and break off easily. Under higher magnification the barbules are seen to possess normal hooks, unless—as is frequently the case—they have broken off.

Many squabs which died young were not classified as normal or scraggly and condition of the skin was only occasionally recorded and rarely relied upon as a basis for classification. Many observations, however, suggest that the scraggly character predisposes a squab to succumb more readily to unfavorable conditions than does the normal. Though proper evaluation of the relative mortality is not possible it is very probable that the moderate deficiency of scraggly individuals in the ratios (Table I) is to be accounted for on this basis.

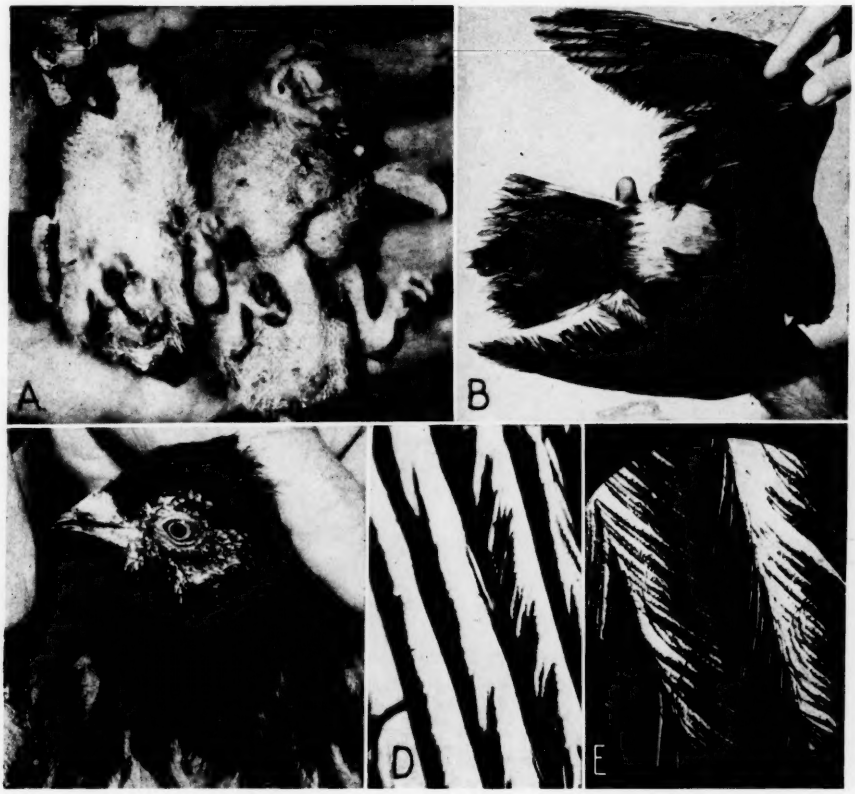
The only other known plumage defect of pigeons resembling scraggly is "silky." The latter is different from scraggly not only in microscopic features and skin texture but in inheritance, as it is dominant to normal.[1] Other plumage defects which are recessive in inheritance are clearly different from scraggly in appearance.[6] No evidences of linkage between scraggly and certain other known factors have appeared (ataxia, piebald coloration, red color, checker or bar patterns).

Ataxia

The original case of ataxia, ♀ *151*, arose in the autumn of 1914 from related parents of Homer stock. The exact degree of relationship of the parents is unknown. Ataxic *151* was first mated with a male Wild Rock (*C-B9*) of stock imported from islands north of Scotland. Later, as already noted, this female was mated with scraggly ♂ *126* (a relative) in order that the heredity of these two characters might be studied together. A preliminary report on the origin and inheritance of ataxia in pigeons was given in 1918 by the senior author.[7] The conclusion reached at that time was that the trait behaved like a Mendelian recessive, with some irregularities. Hoshino[2] provided the neurological data which definitely established the fact that this defect, as it was observed in four F_2 and F_3 descendants of ♀ *151*, is ataxia.

Koch and Riddle[3,4] made comparative studies on the brains of ataxic and normal pigeons of the same family. Weights of the brains, especially of the cerebellum and medulla, averaged somewhat smaller in the ataxics, though variability was great. Chemical differences between the ataxic and the normal type brains consisted in decreased percentages of lipoids, phosphatides, and cholesterol in the ataxics. These differences were especially marked in the cerebellum-medulla segment of the brain. It was concluded that the brains of the ataxics were chemically less differentiated—more infantile—and that in them myelin develops more slowly or less extensively than in normals.

Hoshino made a thorough neurological study of the brains and spinal cords of four markedly (but unequally) ataxic pigeons of this strain and compared them with normal relatives. Many histological defects, varying somewhat in degree in the different specimens, were observed in the ataxic brains and cords. Hoshino stated: "The small size of the brain and spinal cord in all affected birds can be recognized at a glance without any hesitation, especially in the spinal cord, cerebellum, and medulla oblongata. . . . The changes in the central nervous system in the affected pigeons may be regarded as a hypoplasia or developmental



SCRAGGLY PIGEONS

Figure 5

A—Normal squab (right) and scraggly squab four days old, showing scaly, ichthyotic skin, with the plumage slow in developing. Some feathers grow under the skin and give a bumpy appearance. The crop of the scraggly squab is tightly corseted by the stiff skin. *B*—Adult scraggly pigeon showing defective plumage. Scraggly birds cannot fly because the defective feathers do not make a workable airfoil. *C*—Detail of head of adult scraggly pigeon showing bare area around the eyes. *D*—Microphotograph of scraggly feather structure. The barbules are present but mostly fail to spread and interlock. They are brittle and break off easily. *E*—Microphotograph of normal feather for comparison.

inhibition in the proprioceptive system, part of the motor system, and some structures connecting the medulla and cerebellum, occurring during growth with scarcely any definite degeneration." Although many regions which he examined showed reduction in myelination and also in fiber number, there seemed to be no obvious explanation as to why certain ones were unaffected, while others were markedly reduced. His birds showed the same head nystagmus responses to rotation as do normal birds. It was concluded that the semicircular canals were unaffected. Eyesight and hearing also appeared normal. Hoshino pointed out that the condition in pigeons has features resembling those found in both Friedrich's ataxia and Marie's ataxia in man; these latter he regarded as two different aspects or grades of the same basic defect. Ataxia in man has been attributed to dominant hereditary factors.[6] An ataxia-like but fatal con-

ATAXIC PIGEON
Figure 6

An ataxic bird sits balanced on the tail in a characteristic posture. Gait and flight are very irregular and there are persistent nodding movements of the head.

dition in rabbits is inherited as a recessive unit according to Sawin.[8]

For the present study only behavior is considered as a basis for considering a bird ataxic. As a rule this is satisfactory. Ataxic specimens may show a variety of incoordinations, especially when activity is increased by fear, excitement, or in directed movement. The more usual manifestations of the disorder are nodding of the head, or nodding and swaying of the head and neck, unsteady gait, tipping backwards or forwards, irregular flight, and sitting on the tail (Figure 6). Occasionally a bird may fall on its side or somersault. Ataxic specimens seldom are able to stand still on a perch and they prefer the ground or a flat ledge.

The greatest source of difficulty in the genetic study of ataxia is classification. Not only are there different grades of the condition in different individuals, but the same individual may sometimes vary with age. The original ataxic female was markedly ataxic when immature, first showing symptoms before weaning age, but when mature she was much less affected. The reverse trend has been observed in some specimens. A bird now (1942) nine years old seems practically normal, and was considered normal when immature, but ataxic for an intermediate period of two or three years. A few cases are persistently borderline, being classed as normal on casual observation, and "very slightly incoordinated" on careful study. And, though intrinsic variability is bad enough, the picture is further clouded by occasional cases of other disease (vertigo, myoclonus), involving disturbances of movement or behavior, which might be mistaken for ataxia. On the other hand, anything remotely similar to ataxia in our breeds or families of pigeons is extremely rare, and the successful propagation of ataxia in this family for 28 years is in itself evidence that there is an inherited integral basis for it.

In the present genetic analysis we consider any bird in the records normal unless it was otherwise designated, but as ataxic if it was designated as showing ataxia in any degree whatsoever. Possibility of error is unavoidable here since some records were made during a 28-year period by various assistants as well as by the authors, and some of the classifications were not made in a systematic manner. Even greater error, however, would result if the present analysis attempted to split the ataxics into subgroups. A further study specially directed to that end might provide clear-cut classifications, and perhaps show that more than one grade or degree of ataxia is genetically determined.

The F_1 from the original ataxic female and the wild Rock male consisted of three young, all normal. Two were mated to produce an F_2. Of the 27 F_2 young, four were not classified (died too young), six were ataxic, and 17 normal. This result would fit perfectly the explanation that ataxia is a simple Mendelian recessive trait.

The other mating of the original ataxic female, with the scraggly male, gave 30 classifiable young, of which 27 were normal and three ataxic (two slight, one unquestionable). It is of course not easy to explain such a ratio; the fact that these two parents were probably related is perhaps important. Seven matings between these normal F^1's were made. Every one of these matings produced one or more ataxic F_2's, the totals being 54 normals and 21 ataxics. Ataxics of both sexes are represented. A simple

recessive Mendelian factor, not sex-linked, will again account for the results, if we ignore the three ataxics (slight) in F_1.

Four other matings of normal F_1 sibs from later crosses of ataxic × normal were made. Only one of these failed to produce an ataxic offspring, and it produced but a single squab. The totals are 20 normals and 11 ataxics. Eight matings of ataxics with normal F_1 birds from later crosses of ataxic × normal were also made. These gave few offspring, probably because reproduction is difficult for many ataxic birds. The totals here are 16 normals and 13 ataxics. Again the results are in accord with the postulated mode of inheritance.

An analysis of the total data, without reference to particular generations gives results shown in Table II. The matings of ataxic birds *inter se* seem to yield results in flagrant disagreement with the above Mendelian hypothesis. On breaking up the data it is found that one mating produced most of the difficulty. Of 11 classified young, only one showed any sign of ataxia—the nine-year-old male referred to above. On the other hand, six of the matings gave only ataxic young (one possible exception was called a "questionable normal"), 25 in all. Three additional matings gave mixed results, totaling 15 ataxics and four normals.

Probably the simplest assumption that may be made to account for the irregularities observed is that ataxia, although mono-genic, is occasionally able to dominate over its normal allele and that some heterozygous individuals show some (probably mild) ataxic symptoms. Perhaps the "borderline cases" are of this constitution. No valid tests of this assumption are now available, but it seems more logical than to assume multiple-factor inheritance or chromosomal aberration.

Conclusions

The mutant plumage and skin abnormality "scraggly" is characterized by crusty overgrowth (ichthyosis) of the skin, and failure of the barbules to assume normal orientation in the feather. Affected squabs appear to require additional warmth and care to prevent higher than normal mortality. Simple recessive inheritance is demonstrated, and the factor symbol *sc* is proposed.

The mutant "ataxia," characterized by various degrees of defective development of specific areas and nerve tracts in the brain and spinal cord and by associated incoordination of movements, appears to be governed by a single Mendelian factor which is generally recessive, but irregular—occasionally dominant. The symbol *at* is proposed for it.

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TABLE I. Inheritance of scraggly plumage in pigeons

Type of mating	No. of matings	No. of eggs	No. of young: Scraggly	Unclassifiable	Normal
Scraggly x scraggly	11	199	58	34	0
Expectation			58	--	0
Scraggly x pure normal*	9	161	0	7	71
Expectation			0	--	71
Scraggly x heterozygous normal†	22	446	55	43	69
Expectation			(69 or† 61		(69 or 61
Heterozygous normal x heterozygous nor.†	47	1334	110	180	384
Expectation			(128 or† 123.5		(384 or 390.5

* A "pure normal" is a bird from unrelated stock, or from the same stock provided neither of its parents was scraggly. In the latter case the chance of misclassification is at most 50%, and probably much less since no scraggly young were produced.
† A "heterozygous normal" is a bird with a scraggly parent, or which itself produced scraggly young.
‡ Alternative ratios show expectation on the basis of an assumed higher mortality in the scraggly class (above), or without such an assumption (below).

TABLE II. Inheritance of ataxia in pigeons

Type of mating	No. of matings	No. of eggs	No. of young: Ataxia	Unclassifiable	Normal
Ataxic x ataxic	10	272	41	48	14
Expectation			55	--	0
Ataxic x pure* normal	43	785	4†	73	172
Expectation			0	--	176
Ataxic x heterozygous normal‡	46	412	41	78	43
Expectation			42		42
Heterozyg. normal‡ x heteroz. normal	50	1321	93	203	306
Expectation			100		300
Heterozyg.(?) normal x pure normal	21	491	2	47	154
Expectation			0		156

* "Pure normal" designates a bird from unrelated stock, or from the same stock provided neither of its parents was ataxic, and that it produced no ataxic offspring. The original mating (birds not unrelated), however, is included.
† One was probably a case of vertigo (sudden onset, death 5 days later), the others from the original mating.
‡ A "heterozygous normal" is a bird with an ataxic parent or one which produced ataxic young.

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GENETICS OF THE MOUSE

IT is refreshing and encouraging to find that in spite of abnormal and disrupting war conditions, a young British scientist has pioneered in producing the first really comprehensive and adequate work on the genetics of a mammalian species.

A survey of our knowledge of the genetics of the mouse makes a showing which will be surprising even to those in the field. We hope it will be something of an eye-opener to those engaged in research in experimental medicine or in other genetic investigation.

Grüneberg's work* impresses the reader as being the result of a sincere and intelligent absorption in the material which he describes. His attitude on unproven or controversial issues is judicial and non-cynical throughout. A confidence in the future advances to be expected gives a strong undertone of intellectual optimism which is a welcome contrast to a too highly developed sense of "constructive" criticism so often found in similar situations.

As an example of the author's somewhat unusual sense of proportion is his attitude towards the already published book on *The Biology of the Laboratory Mouse*.† This work he accepts as sufficiently adequate in its field, and he thereupon focuses the major part of his attention on reviewing, recording and discussing the genetic phenomena as such.

His interpretation of genetics is properly broad and constructive. It recognizes the peculiar fitness of mammalian material for investigating the relation of gene to character. Physiological implications are held to be of vital importance—as indeed they should be.

In the early chapters the author shows by his evaluation of such terms as "qualitative" versus "quantitative," and "morphological" versus "physiological" a sane and logical philosophy likely to engender well-deserved confidence on the part of the reader.

A brief introduction and discussion of the taxonomic position of the mouse are followed by a statement of the more important facts of reproduction and growth. All of these occupy only some twenty pages. The main body of the book, a section of over 250 pages, is entitled: "The Inherited Differences." It is comprehensive and up-to-date.

Structure Differences

Under the first main sub-division, that of morphology, the so-called "qualitative differences" are first considered. These are divided according to the location in which they express themselves. An excellent system of cross references prevents too rigid a classification and is of great assistance to the reader. The locations listed are, in order: the integument, endocrine organs, brain and sense organs, blood and blood-forming organs, skeleton, alimentary tract and urogenital system.

There is an interesting description of the ten recorded groups of color genes with brief general discussion of the determination of hair pigments and of color mosaics. Topics of this sort are treated

*Gruneberg, Hans. *Genetics of the Mouse.* Pp. 412. $7.00. Cambridge University Press and Macmillan Co., New York. 1943.

†*The Biology of the Laboratory Mouse,* by the staff of the Roscoe B. Jackson Memorial Laboratory, G. D. Snell, Editor. The Blakiston Company, Philadelphia. 1941.

in a way likely to stimulate an appreciation of genetic values by those primarily interested perhaps in biochemistry or morphogenesis. This catholic attitude towards bordering sciences is an excellent feature throughout the book.

The genetics of spotting, always to some degree a "no man's land" for well meaning geneticists, is adequately considered and well illustrated. The need for much more study is properly emphasized. Hair texture, with excellent summaries of the known histo-anatomical basis for the various genetic types completes the section on the integument.

Endocrine organs are next considered. Here chief emphasis is placed on the better known genetic material; namely, dwarfism due to anterior pituitary deficiency. The section is quite as stimulating in its suggestions of topics offering opportunity for further study as it is satisfactory in the description and analysis of dwarfism.

The consideration of brain and sense organs includes absence of corpus callosium, pseudencephaly, hydrocephaly (in two forms) and the various manifestations of nervous instability as observed in the "shaker," "waltzer," "jerker" and "circler" series. There is also adequate discussion of various ear abnormalities and of a series of eye malformations including anopthalmia, rodless retina, cataract, "open eyelids" and a number of rarer and less well analyzed characters.

There then follow the anomalies of the blood and blood-forming organs including "macrocytic anaemia" (dominant spotting in its various allelomorphic forms). This character is one of those extensively studied by Grűneberg himself. "Flexed tail," a coincidental manifestation of a condition described as "siderocyte anaemia," is considered in a thoroughly scientific and comprehensive manner. A series of abnormalities as yet incompletely analyzed completes this section.

One of the most interesting groups of genetic characters is that of the mouse skeleton. Beginning with "gray-lethal," Grűneberg's own discovery, the anatomy, histology, origin and biological nature of a number of variations are carefully discussed. Among these is the short-tailed (brachyury) series extensively and skilfully investigated by Dunn and his associates. The different interactions of the alleles are described, as is also the up-to-now unique evidence of gametic selection in certain types of male gametogenesis. The tail has proven to be an extraordinarily variable structure in mice and the genetics of "kinky-tail," "pig-tail" and similar anomalies remains in need of further study. Danforth's "posterior reduplication," a bizarre and irregular abnormality, irregularities in rib number, "absence of the tibia," "polydactylism," "oligodactylism," "twisted nose," "parted frontals" and "interfrontal" are also briefly covered. They are further examples of characters needing much more observation and investigation.

"Harelip and cleft palate," "absence of palatal ridges," "stomach lesions" and lethal jaw abnormalities form a small group of characters affecting the alimentary tract. The urogenital system is represented by abnormalities of the renal system, by a type of imperforate vagina and by irregularities in the mammary glands and secondary sex characters. All of these characters are as yet incompletely analyzed.

The treatment of qualitative differences is brought to a close by a brief general discussion of principles involved in the time and place of origin of the various genetic characters in this group. The nature of the mutational process is also discussed in an interesting and helpful way. The type of tabular analysis used by Grűneberg shows his naturally logical and orderly way of approaching the material. In the more than 200 pages given to the consideration of qualitative characters he has done a masterful piece of work with a minimum of complex and confusing steps.

Number Differences

By its relative length and essentially general statements the shorter section on "Quantitative Differences" confesses the comparatively imperfect stage of our

knowledge in this field. Litter size and body weight are the most clearly understood and most thoroughly investigated. Other size criteria and the number of tail rings are still very imperfectly analyzed from a genetic point of view.

Physiological differences are sub-divided into "Serology" where some analysis has been possible and a series of "miscellaneous" physiological differences such as xanthine oxidase activity in the liver," "water requirements," "hyperglycaemia," "temperature characteristics of breathing" and a strange tendency towards "whisker eating" by some animals.

A section on the "Resistance to Infectious Diseases" emphasizes very wisely the diphasic nature of the problem. Host and infective agent are both potential variables. It is also true that the very nature of the process of immunity makes it difficult to separate the truly genetic elements from those that are not. Many environmental factors may enter in to complicate matters further. Six relatively common infective agents are discussed in general terms with adequate references to the literature. Virus diseases and specificity of resistance are also briefly considered.

As might well be expected, the genetic situation becomes still more ephemeral when the author discusses "Behaviour." This does not mean that this phase is unimportant; rather that investigators have, very naturally, elected to place more emphasis on the study of the simpler physical differences.

A compact and helpful chapter on "Chromosomal Inheritance" gives essential data on cytology and on both independent and linked genes. This is in convenient form for ready reference.

The final chapter in the main body of the book is on induced genetic changes with special reference to x-rays and other irradiation. Alcohol treatment and hormone treatment are also discussed.

An appendix on the "Genetics of Cancer in Mice" by the reviewer and Dr. P. A. Gorer takes up the main developments with transplanted, induced and spontaneous tumors. A second appendix contains information on the "Keeping and Breeding of Mice for Genetical Experiments."

A bibliography, conveniently arranged by chapters and by genetic characters, contains more than 1,100 references. The index is adequate and is subdivided under authors and subjects.

It seems to the reviewer that Dr. Grüneberg's book is a complete and scholarly piece of work. It is also of great value to the investigator and will remain an excellent reference volume. It is, as before mentioned, the first attempt at covering in detail the genetics of an experimental laboratory mammal. As such it sets a high level of excellence which does credit to its author and provides a challenging standard to measure future works by. It is difficult to see how anyone using mice as experimental material can afford to be without a copy.

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RACE DIFFERENCES IN INTELLIGENCE

IT has always been difficult, if not impossible, to conduct certain types of studies with respect to the relative distribution of physical and mental traits in the United States according to race. This is due primarily to the failure to classify the general white population according to race. The absence of such census material makes it impossible to compute rates essential to such an analysis. Perhaps for this reason, it will be found that the earlier writers rarely, if ever, commented upon the frequency of mental deficiency among the several racial aggregates in the United States.

Interest in the subject developed as a result of the application of intelligence tests to selected populations of varying racial and national origins. As a result of such examinations, it became evident that the frequency distributions of intelligence, as measured by modern intelligence scales, varied from group to group. The most obvious of such comparisons is that between Negroes and whites. An immense literature has developed about this subject, adequate summaries of which has been prepared, among others, by Klineberg and by Pintner. The lat-

(*Continued on page* 192)

BULLDOG AND HAIRLESS CALVES

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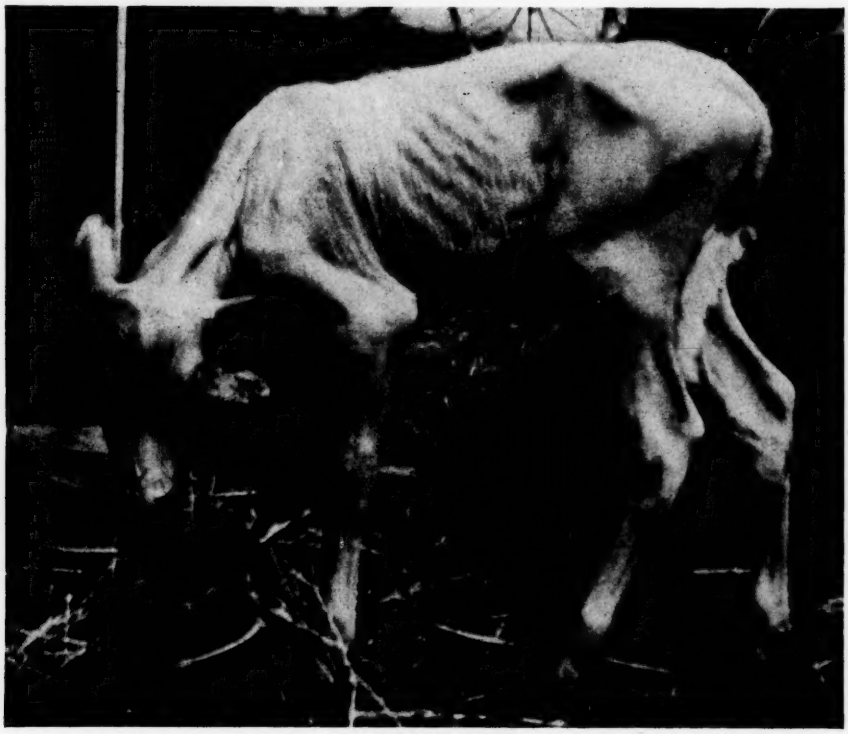


HAIRLESS CALF

Figure 7

At birth there were a few hairs under the lower jaw and the lower third of the tail had considerable hair. The rest of the body was covered with a sparse fuzz by the time the calf was slaughtered at seven months of age. The defect appears to be inherited as a recessive characteristic.

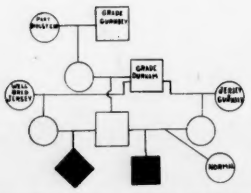
THE problem of abnormal calves is one of considerable economic import. Mohr and Wriedt,[6] Eaton,[4] Heizer and Marshall,[5] have summarized some of the known abnormalities.

The cases reported in this paper were found, not by a careful survey, but through hearing by chance of reported cases which appeared in herds in the neighborhood of Berea, Ohio.

Hairlessness

The condition of hairlessness has received considerable attention in numerous animals, cattle, cats, dogs, goats, mice, rabbits, rats, and swine. These conditions are not considered parallel or identical in the opinion of Wilder et al[10] since the expression of the character differs widely in phenotypic expression and degree of dominance or recessiveness.

Hairlessness in cattle is recorded by Mohr and Wriedt,[6] Craft and Blizzard,[3] Regan, Mead and Gregory,[9] and Wipprecht and Horlacher.[11] They consider the character to be a Mendelian recessive and all cases are reported as lethal except the one by Regan, Mead and



PEDIGREE OF HAIRLESSNESS

Figure 8

The inbreeding in the ancestry of the two hairless calves suggest that a recessive factor is responsible for the condition.

BULLDOG CALVES

Figure 9

Three related calves from inbred matings showed a remarkably similar constellation of defects. Short upper jaw, bulging eyes, notched ears, and gnarled legs were among the more striking deviations from normal found in these calves.

Gregory which they call a sub-lethal.

The hairlessness here reported was found within a rather short distance of the "bulldog calves," which may emphasize the probable commonness of abnormalities in cattle. The hairless calf was an otherwise perfect full term animal. Its body was hairless except for a few hairs under its lower jaw and the last third of its tail was rather well haired at the time of birth. The animal was healthy in all respects except that it could not withstand the sun and was subject to constant irritation by flies. The owner made a tightly fitted cover for the animal which seemed to protect it rather well.

The animal was kept by the owner until about seven months old and then killed for home consumption. The animal developed a sparce covering of fuzzy hair over most of its body before it was killed. Inquiry elicited rumors of one other similar calf born in the vicinity. This case was investigated and the owner reported it as identical with the case described. The animal had been sold to a slaughter house when four weeks old, when it was said to weigh 150 pounds.

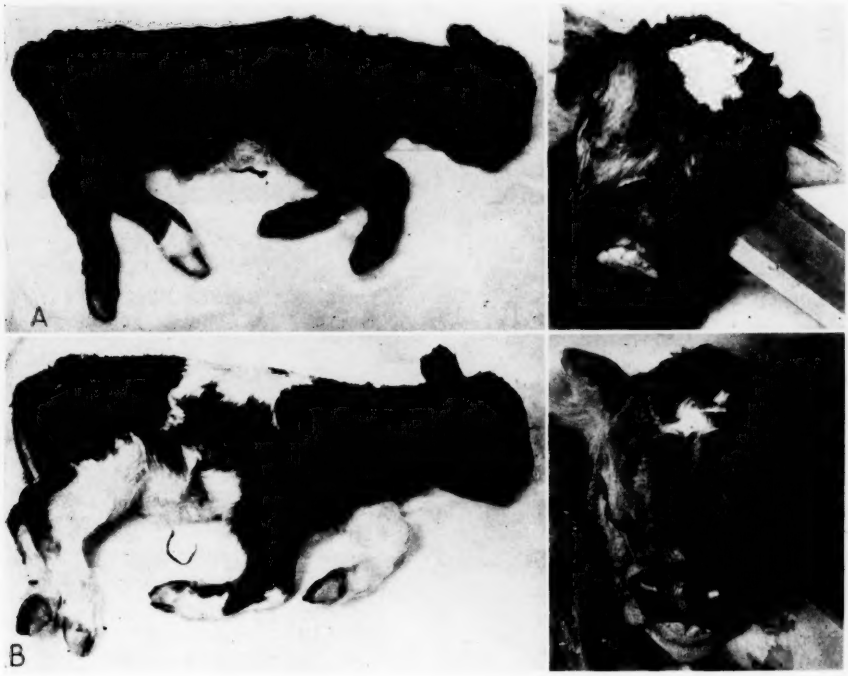
Figure 8 illustrates the degree of inbreeding and Figure 7 the appearance of a hairless calf. Considering the normal appearance of the sire, dam, and sisters of this animal, we may postulate that the inheritance of the defect is a simple Mendelian recessive.

Bulldog Calves

The bulldog condition here reported is apparently quite similar to uses earlier described by Mohr and Wriedt,[8] Carmichael,[1] and Punnett.[8] The first case noted of the "bulldog" condition (Figure 9) was produced from a mating of an excellent grade Jersey cow to a thoroughbred Jersey sire eligible to registration. This abnormal calf was not considered especially significant by the author and little more than a careful examination of the animal was made.

The matter took on a new significance when about a year later in the same neighborhood, I learned of another peculiar calf (Figure 10*A*). This animal was carefully examined. Two months later the same farmer reported another bulldog (Figure 10*B*).

The three calves were remarkably similar with a pronounced under bite due to the shortened maxillae, thus with buccal exposure. The eyes were bulged to the extent that the lids could not be closed, ears were notched quite similar to "notched ear," a dominant mutation reported in Ayshire cattle. The legs were gnarled to the extent that the calves could not support themselves. All were without tails and were hermaphrodites. One of the calves was carefully dissected. The four teats were symmetrically placed with a small amount of mammary tissue immediately under them. The two scro-



DETAILS OF BULLDOG CALVES

Figure 10

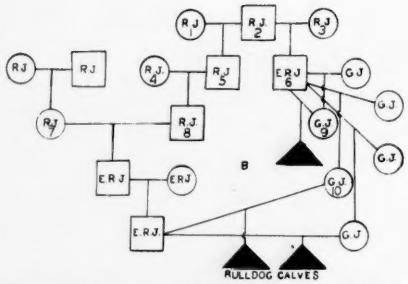
Body view and heads of two of the bulldog calves shown in the pedigree. Note the similarity in appearance.

tal sacs were behind the last pair of symmetrically placed teats, but were empty. The right testis was in the abdominal cavity—the left one was out in the subcutaneous tissue close to, but not in, the scrotal sac. A vagina and anus were present. Figure 12 diagrammatically illustrates the inter-sexual relationship which is of considerable interest from an embryonic and an anatomical standpoint. All calves were full term and born alive. None of them could possibly have lived without a great deal of artificial assistance and even then survival would have been questionable.

The pedigree (Figure 10) illustrates the relationship of the deformed calves. Animals 1, 2, 3, 4, 5, 7, and 8 are all "pure bred" with notable records. Animal 2 was a high priced animal, a Gold and Silver Medal sire, with numerous daughters in the Register of Merit. It is evident from Figure 10 that the "bulldog" character acts as a typical Mendelian recessive.

There is reason to believe that such abnormalities are probably rather common. In addition it is quite possible that many of the cases of abortion falsely diagnosed as infectious may rightly be of an inherited nature. The above data concern two abnormalities resulting from inbred lines. Inbreeding of cattle by small owners is probably much more intense than is usually supposed. It may be somewhat indirect; but often a neighborhood sire is an animal selected more for his convenience than for breeding qualifications. Often it will be found that he is related to the previous generation of neighborhood stock.

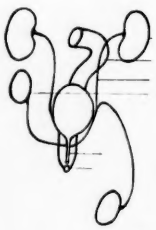
Certainly it must be kept in mind that



RELATIONSHIP OF BULLDOG CALVES

Figure 11

Several pure bred animals with notable records were found among the ancestors of these calves.



MIXED SEX

Figure 12

Diagram of urogenital system of one of the bulldog calves, showing the right gonad in the abdominal cavity and the left in the scrotal sac. All three bulldog calves were similarly affected.

inbreeding is not the *cause* of abnormalities, since it only brings to light undesirable characters in the line. There may be some truth in the "paradox of lethal genes and great sires" as is well illustrated in the case of the bulldog calves here recorded. The maintenance of strains having both advantageous and pathological genes might be economically feasible to the fancy stockman. Certainly it is not a wise policy for the dairyman who deals for the most part in grade stock. To such individuals lethal and abnormal genes are of considerable consequence. A plan of stock purification such as that proposed by Mohr and Wriedt might be highly beneficial to all concerned. But the test of mating sires to 20 of their own daughters is hardly feasible for the farmer and small breeder. The production of sires free from recessive defects and homozygous for high production is a long-range undertaking in which several of the State experiment stations and the U. S. Department of Agriculture are fruitfully cooperating.

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Fifty-Seven Varieties

The phylogenetic siblings of *Drosophila melanogaster* offer unique material for studying the origin of species. Painter's homologizing of the salivary gland chromosomes and the ordinary, garden variety of chromosome makes possible comparative studies of chromosome and genetic phylogeny in the Drosophilæ in a manner still impracticable in any other organism. The publication by the University of Texas of an N.Y.A. project to list and describe the drosophilæ of the Southwest is thus a noteworthy example of how funds expended through the now extinct N.Y.A. could and contribute to the progress of science.*

It happens that there are fifty-seven species of Drosophilæ listed from the State of Texas. The other southwestern states are also included so that the total is much larger than this. Thereare excellent colored plates illustrating seventy-five of the forms described. Chromosome analyses are presented for seventy-one species. The monograph is richly illustrated with drawings of the reproductive systems of the various species described.

*Studies in the Genetics of Drosophila III. by James G. Patterson, Pages 327. Price $2.50. The Drosophilæ of the Southwest. Directed University of Texas, Austin, 1943.

PICTORIAL PRESENTATION OF ANTIGEN AND ANTIBODY RELATIONS

Associated With the "A" Character in the Rabbit

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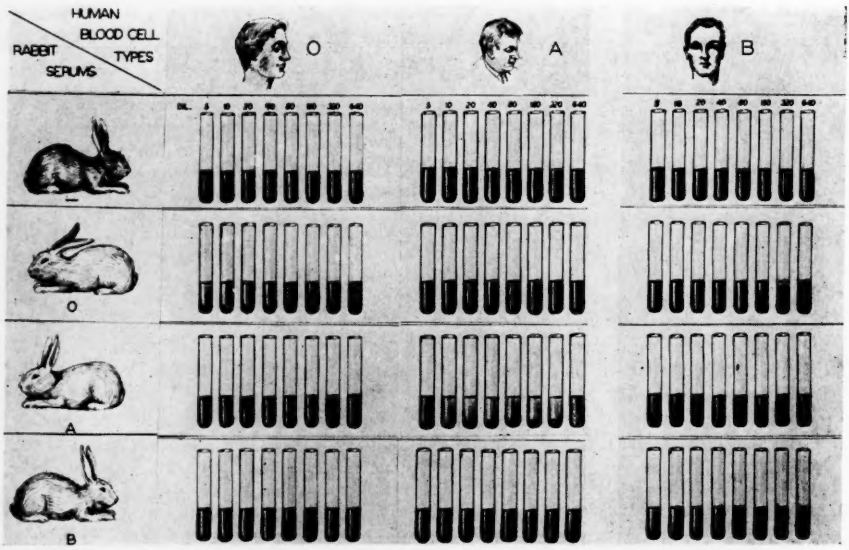
SOME differences in antigen and antibody constitution found in human and animal bloods appear to be characteristic of a particular species. Others are peculiar to individuals and have been generally ascribed by immunologists to constitutional differences.[3] A few antigens at least have been shown by Irwin, et al.,[1,2,4] to result from gene action in hybridization. Comparison of the incidence of individual variations as obtained in the offspring of specific variates with that found in populations selected at random, as for example in the case of Landsteiner and Levine,[5] have in a number of cases indicated that such differences might be inherited without giving precise information as to the nature of that inheritance. In a few cases such as the blood groups in man and in the rabbit sufficiently critical genetic data have been accumulated to be certain that the hereditary transmission is typically mendelian.

Variations of this kind which are inherited are important not only as genetic material but as affording a means of biological analysis and standardization comparable to the standardization of reagents so important in the general field of chemistry. This is particularly important in animals such as the rabbit whose blood is so generally utilized in the preparation of immune serums and in other clinical procedures, and also for various types of serological and biological investigation. Efforts to determine the manner of inheritance of some of these differences have been reported from this laboratory from time to time.[9,10] Concomitant with the progress of these experiments it has become increasingly apparent that the internal patterns involving associations of antigen and antibody which are obtainable within the species may be as numerous and complex as are the external patterns of coat color and influenced in the same way by the interaction of both genetic and environmental forces. The very nature of these variations makes their genetic analysis dependent upon precise qualitative and quantitative methods which are more or less routine in the serological laboratory but which are not so well understood outside. The purpose of the present paper and its accompanying diagrams is to portray the serological manifestations of the *"A"* character in the rabbit and to record further data pertaining to its inheritance.

The Alpha Agglutinins

Presence of an alpha agglutinin may be recognized by the ability of a rabbit serum to agglutinate human erythrocytes of the *A* type, but other serological manifestations may be associated with it as will be described presently. The agglutination reaction is explained on the basis that rabbits whose serum will agglutinate human cells normally possess antibodies for those cells, whereas rabbits whose serum will not agglutinate human cells are lacking in such antibodies. Normal agglutinins can also be demonstrated in some rabbits for human *O* type cells and also for the *B* type. The former, commonly recognized as lacking both the *A* and *B* antigens, possess an antigen specific for the species and some additional antigen.

In testing the serum of a given rabbit for the type or types of human cells which it will agglutinate, serial dilutions of the rabbit serum are prepared in sets ranging from 1:5 to 1:640 as shown in Figure 13. To each set is added 0.5 ml. of a 0.3 per cent suspension of human



TESTING SERUM FOR ABILITY TO AGGLUTINATE HUMAN RED BLOOD CELLS

Figure 13

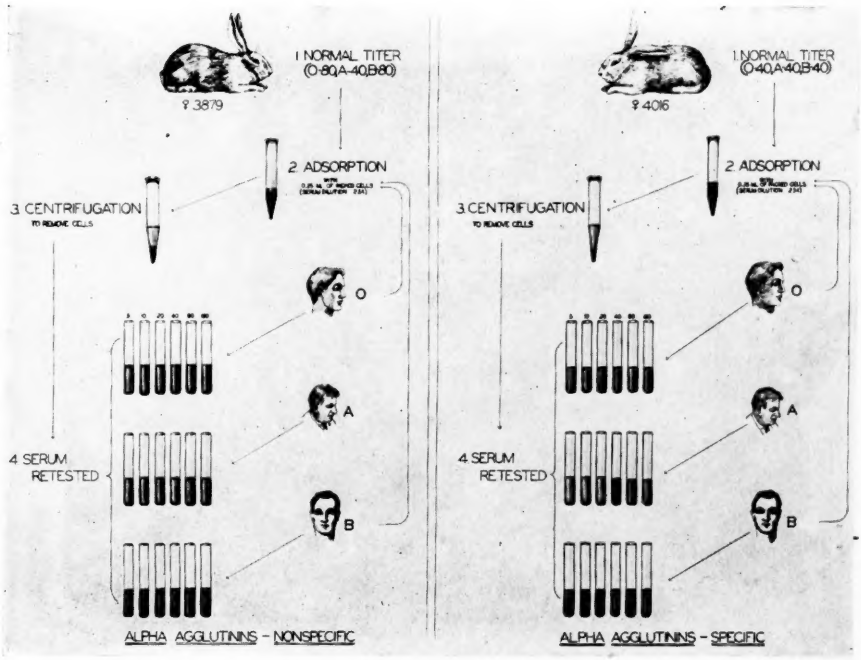
This chart shows the technique of differentiating the interactions between rabbit serums and human blood groups. The test tubes contain rabbit serum progressively diluted in proportions of 1:5 to 1:640 of the rabbit serum. To each of these test tubes is added an equal amount of human red blood cells of the three types shown. It is seen that the first rabbit agglutinates none of these, the second rabbit agglutinates only the human *O* cells, and the third rabbit agglutinates only human *A* cells, and the fourth rabbit agglutinates only *B* cells. Some rabbits agglutinate only one type of human cells and others agglutinate several. The sorting out of the various elements when this is the case is shown in Figure 14.

erythrocytes, in the first set of type *O*, the second of type *A* and the last of type *B*. The degree of agglutination is recorded after two hours' incubation at 37°C. This quantitative procedure is most important because a prozone is occasionally encountered and also because of the temperature relationship between antigen and antibody.[7] For the first rabbit (Figure 13) no reaction is observable for any of the human cell types, (either *O*, *A*, or *B*). In the dilution set of the third rabbit for example, agglutination is clearly manifested in all but the last tube of the series containing the *A* type cells. The reaction is first observed by the clumping of the cells in a manner similar to that partrayed in this JOURNAL by Cumley and Irwin,[2] and finally by the settling out of these cell clumps leaving a clear supernatant liquid.

Rabbit serums have been found which agglutinate only one type of human blood cells, either *O*, *A*, *B* or *AB*. In some exceptional cases agglutination will occur for several or even all cell types. When such cases occur the specificity of the *A* type for example can be demonstrated by the *adsorption* test portrayed in Figure 14. The procedure consists essentially of treating each ml. of serum which has first been diluted 2.5 times with 0.25 ml. of packed cells of the *O* and *B* types at room temperature for 30 minutes. After centrifugation to remove the cells the serum is again tested in the usual manner.

Immune Alpha Agglutinins

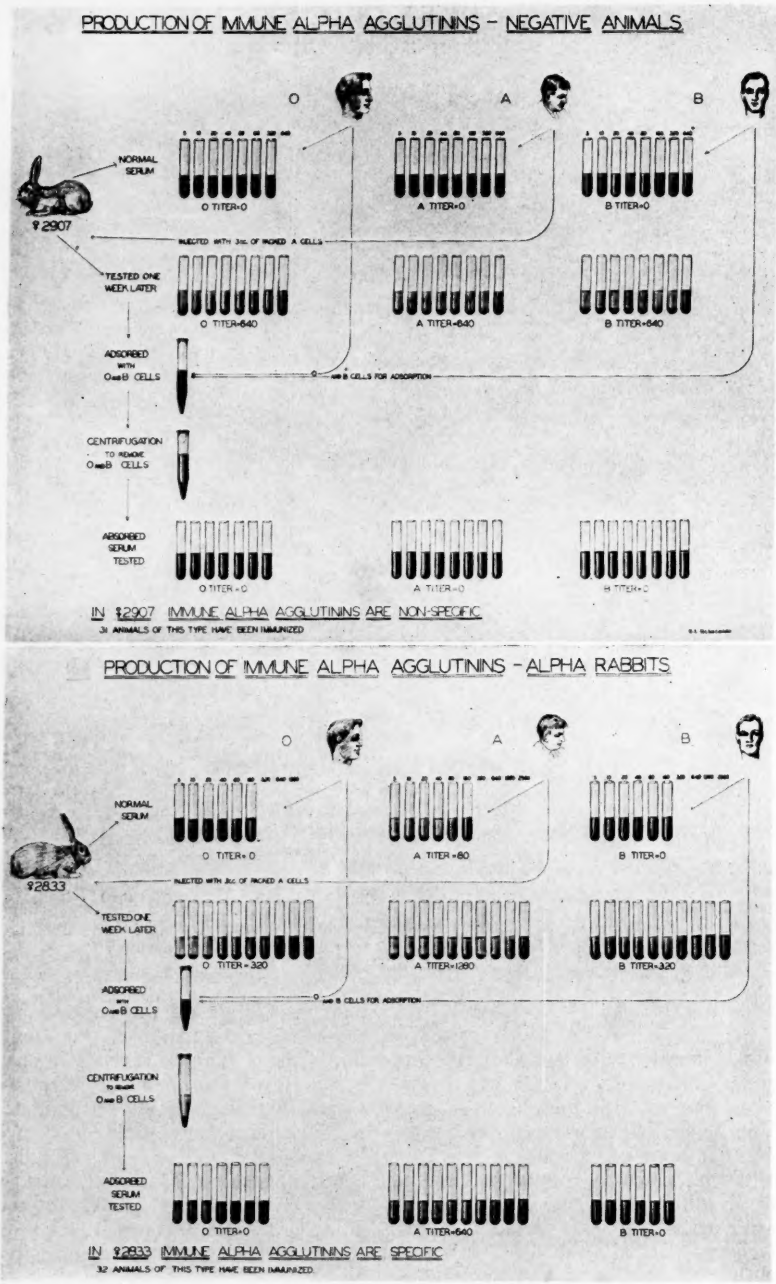
Antibodies found in human and in animal serums may arise in two ways. Those just described, which occur nat-



DIFFERENTIAL ADSORPTION
Figure 14

Rabbits which agglutinate one or more types of human blood cells can be tested for specificity by absorption with human red blood cells of known types. When the serum of rabbit 3879 is absorbed with *O*, and *B* cells, and then tested with *O*, *A*, and *B*, no specific reaction is obtained. When the blood serum of rabbit 4016 is treated in this way a specific reaction for the human *A* blood cells is obtained. In the one place we have a generalized reaction against human red blood cells of any type, and in the other a specific reaction against human *A* antigens. These two types of rabbits react differently when antibodies are formed artificially through injection of human *A* cells. The details of this are shown in Figure 15.

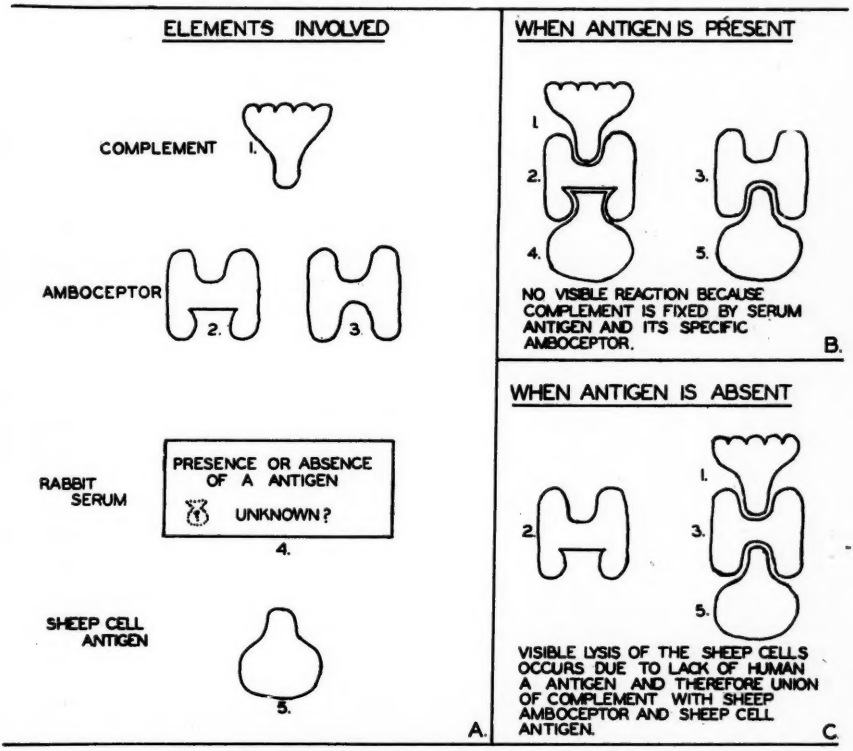
urally, are referred to as normal antibodies; whereas those which arise by artificial immunization are referred to as immune antibodies. There is a well recognized variation in the ability of rabbits in general to produce immune antibodies for human red cells. The reason for this is shown in Figure 15. In the case of rabbit 2907 (upper chart), the agglutination test (top row of tubes) showed no normal antibodies. Injection with human *A* cells produced antibodies for all three types. Such a reaction would be anticipated if, in addition to the *A* antigen, the human cells also possessed some antigen specific to the human species. That the antibodies thus produced are actually of this sort and not of the alpha type can be demonstrated by adsorption of the antiserums with *O* and *B* cells. These are found to remove the alpha agglutinins as completely as those for the *O* and *B* cells. In contrast, rabbit 2833 (lower chart) possessed a natural titer of 80 for normal alpha agglutinins. When it was injected with *A* cells it produced agglutinins for all three types of cells, the titer for *A* being especially high. When this serum was absorbed with *O* and *B* cells, both *O* and *B* agglutinins were removed but the titer for the *A* type cell was not al-



PRODUCTION OF IMMUNE AGGLUTININS

Figure 15

The artificial production of antibodies, referred to as "immune antibodies" is demonstrated in the charts. Rabbits vary greatly in their ability to produce antibodies against the different types of human red cells. These charts show how a negative rabbit (one which has no natural anti-*A* antibody) differs in reaction from a rabbit having natural antibodies to human *A* cells. The ability of an animal to respond to immunization seems to be due to his inherited ability to produce antibodies.



THE COMPLEMENT FIXATION TEST

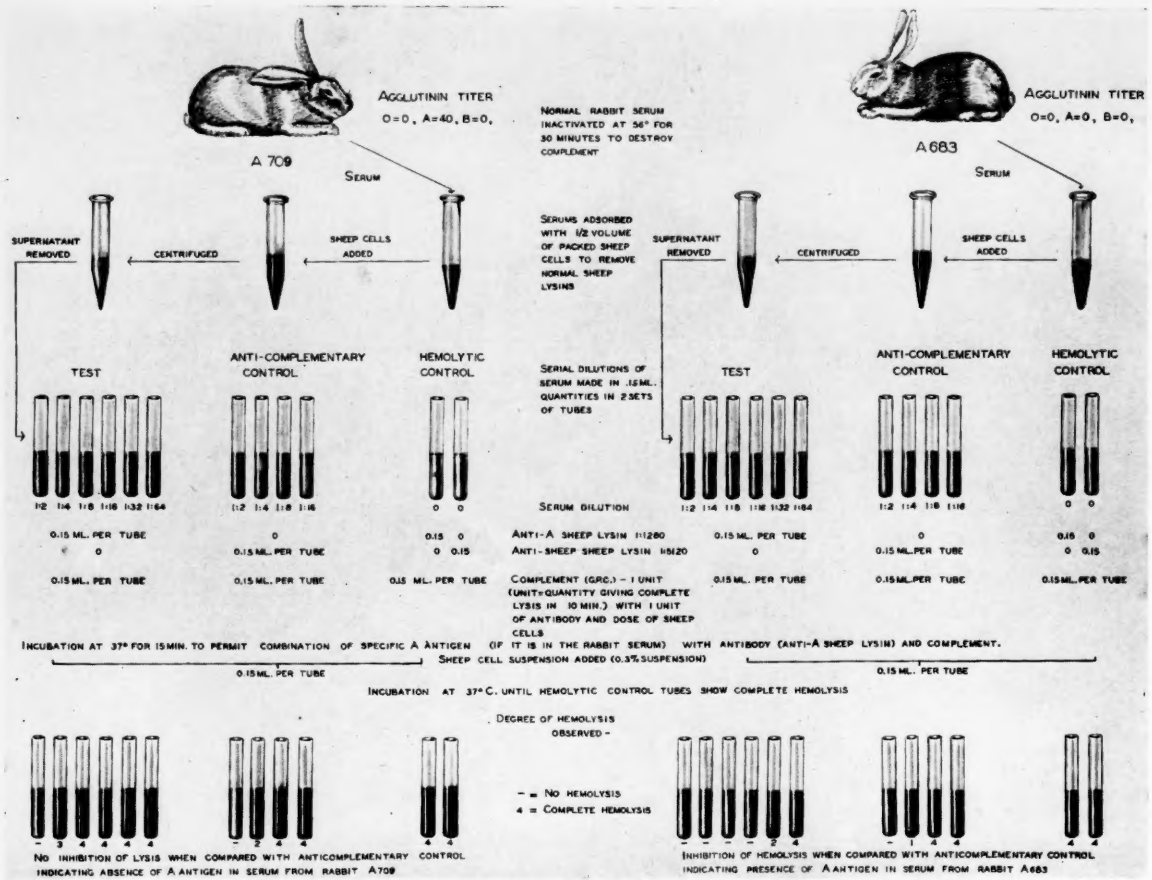
Figure 16

In order to determine the presence or absence of antigen, a modification of the complement fixation test is used. This chart shows the elements which enter into that test. In this chart *1* represents the complement, *2*, represents specific *A* amboceptor in the serum of rabbits immunized with human *A* cells; *3*, a specific sheep amboceptor produced in the same rabbits by the antigen common to human *A* and sheep cells; *4*, the serum of the rabbit being tested for the presence of *A* antigen; and *5*, sheep cells. Whether or not a visible reaction is produced is determined by the presence of the *A* antigen. If this is present the complement is attached to the *A* amboceptor and no reaction occurs. Further details of this test are shown in Figure 17.

tered. This shows that the animals which normally possess some *A* antibodies are the animals which respond to immunization. Thus the presence of such agglutinins is an indication of the ability of an animal to respond to immunization. This ability is an interesting manifestation of the same character and in exceptional cases is useful in differentiation of the type and for other specific purposes, but in itself is not a practical character for genetic study because of the large number of human cells required.

The Double Complement Fixation Test

As soon as the genetic analysis of the distribution of alpha agglutinins in rabbit serums was well advanced, it became apparent that the numbers of animals with alpha agglutinins in both the backcross and F_2 populations was slightly in excess of expectation upon the basis of a single mendelian unit. Various authors have observed that there is an almost regular reciprocal relationship between the presence of *A* antigens and antibodies in rabbit serums. Exceptions


AGGLUTININ TITER
O=0, A=40, B=0,
A 709
SERUM
SUPERNATANT REMOVED
CENTRIFUGED
SHEEP CELLS ADDED
TEST
ANTI-COMPLEMENTARY CONTROL
HEMOLYTIC CONTROL
1:2 1:4 1:8 1:16 1:32 1:64
1:2 1:4 1:8 1:16
0.15 ML. PER TUBE
0
0.15 ML. PER TUBE
0.15 ML. PER TUBE
NORMAL RABBIT SERUM INACTIVATED AT 56° FOR 30 MINUTES TO DESTROY COMPLEMENT
SERUMS ADSORBED WITH 1/2 VOLUME OF PACKED SHEEP CELLS TO REMOVE NORMAL SHEEP LYSINS
SERIAL DILUTIONS OF SERUM MADE IN .15 ML. QUANTITIES IN 2 SETS OF TUBES
SERUM DILUTION
ANTI-A SHEEP LYSIN 1:1280
ANTI-SHEEP SHEEP LYSIN 1:520
COMPLEMENT (GPC.) - 1 UNIT (UNIT=QUANTITY GIVING COMPLETE LYSIS IN 10 MIN.) WITH 1 UNIT OF ANTIBODY AND DOSE OF SHEEP CELLS
INCUBATION AT 37° FOR 15 MIN. TO PERMIT COMBINATION OF SPECIFIC A ANTIGEN (IF IT IS IN THE RABBIT SERUM) WITH ANTIBODY (ANTI-A SHEEP LYSIN) AND COMPLEMENT.
SHEEP CELL SUSPENSION ADDED (0.3% SUSPENSION)
0.15 ML. PER TUBE
INCUBATION AT 37° C. UNTIL HEMOLYTIC CONTROL TUBES SHOW COMPLETE HEMOLYSIS
DEGREE OF HEMOLYSIS OBSERVED -
- = NO HEMOLYSIS
4 = COMPLETE HEMOLYSIS
NO INHIBITION OF LYSIS WHEN COMPARED WITH ANTICOMPLEMENTARY CONTROL INDICATING ABSENCE OF A ANTIGEN IN SERUM FROM RABBIT A709
INHIBITION OF HEMOLYSIS WHEN COMPARED WITH ANTICOMPLEMENTARY CONTROL INDICATING PRESENCE OF A ANTIGEN IN SERUM FROM RABBIT A683
A683
AGGLUTININ TITER
O=0, A=0, B=0,

DETAILS OF TEST

Figure 17

By the complement fixation tests described it is shown that rabbit A709 does not have an *A* antigen in its serum, while rabbit A683 does. See text (page 185) and Figure 16 for further description of this chart.

to this rule are of two kinds: (1) animals which lack alpha agglutinins, possess the *A* antigen and at the same time are capable of producing specific immune alpha agglutinins of a low titer (These animals also differ from normal alpha rabbits, homozygous for the recessive *an* gene, in other qualitative ways.); (2) rabbits possessing both specific alpha agglutinins and the *A* antigen. The first type is not at present of genetic importance. The latter type, however, constitutes a group of *irregular* alpha rabbits which are definitely responsible for the aberrant ratios mentioned above.

Since both the normal and irregular alpha agglutinins are specific and serologically indistinguishable by the ordinary methods of differentiation (agglutination and adsorption), and since immunization is impracticable, the next best procedure is a test for the reciprocally associated manifestation, namely the *A* antigen, the specificity of which can be determined by a modification of the well-known complement fixation test.

Due to the fact that the *A* antigen here is an unformed protein in the serum, the usual three factor system of the complement fixation test (antigen, amboceptor, complement) does not result in a visibly manifest lytic reaction, as would be the case if the antigen were a formed protein such as erythrocytes. The presence or absence of this unformed antigen can be quite as effectively demonstrated, however, by employing a *double complement fixation* or "inhibition" test. In so doing, advantage is taken of two facts. First, normal rabbit complement which is variable in quantity can be easily destroyed by heating to 55°C. and can be replaced in the three factor system by controlled amounts of guinea pig complement. Second, the human *A* cell contains, in addition to specific *A* antigen, another antigen which is also present in sheep cells. Thus, a rabbit immunized with human *A* cells produces not only *A* amboceptor but also a certain sheep amboceptor (anti-*A* sheep lysin, Figure 17). The test for the *A* antigen thus may be set up as diagrammed in Figure 16*A* in which 1 represents the complement; 2, the specific *A* amboceptor in the serum of the rabbit immunized with human *A* cells; 3, specific sheep amboceptor produced in the same rabbit by the antigen common to human *A* and sheep cells; 4, the serum of the rabbit being tested for the presence of the *A* antigen in the serum; and 5, sheep cells. When 1, 2 and 4 are placed together (Figure 16*B*) if 4 contains *A* antigen it will unite with 2 (*A* amboceptor); 2 and 4 form an immunologically specific complex and the complement will be completely fixed. If after a 15 minute interval sheep cells (5), are added, 3 will unite with 5 but there will be no hemolysis of the sheep cells since no complement remains free to activate this system. On the other hand, had 4 lacked *A* antigen (Figure 16*C*) the first reaction would not have taken place and complement would have been free to react on the second (sheep cell) system.

Typical examples of the reactions occurring in a rabbit possessing *A* antigen and in one which does not are shown in Figure 17.* Emphasis, however, should be placed upon the fact that the success of these tests depends upon the accurate quantitative determination of the different units of complement, amboceptor and antigens used in the tests and a proper appreciation of the anti-complementary and hemolytic controls.

In the case of rabbit A709 (Figure 17), it will be noted that there has been complete inhibition of hemolysis in the 1:2 dilution and slight inhibition in the 1:4 dilution in both the test for *A* antigen and in the anticomplementary control of the rabbit serum. Since the test and control reacted alike it is apparent that inhibition of hemolysis in both cases resulted from the anticomplementary reaction of the serum showing that *A* antigen was not present in the serum of rab-

*Also included, are the anti-complementary control of the rabbit serum and the hemolytic controls of the sheep amboceptor in the rabbit anti-*A* serum and the sheep amboceptor in the rabbit anti-sheep serum, the details of which need not be described here.

bit A709. In the case of rabbit A683 the anticomplementary control showed nonspecific adsorption of the complement up to a 1:4 dilution; whereas in the test, complement was fixed up to a 1:32 dilution showing conclusively that this rabbit possessed *A* antigen in the serum. It will be noted that the anticomplementary reactions of the serums of rabbits A709 and A683 were the same. This is typical for most rabbits which shows the importance of the anticomplementary control. The hemolytic controls are essential, since the unit of complement necessary to give complete hemolysis of the sheep cell antigen amboceptor complex established at one time, may within an hour or two be insufficient to give complement hemolysis due to the rapid deterioration of complement when used in dilution.

Inheritance of the *A* Character

Additional evidence on the inheritance of the *A* character accumulated in part by H. Sleeper and reported in an unpublished Master's thesis are shown in Table I. In brief they substantiate the original interpretation, that the presence of the *A* character (*An*), being negative for alpha agglutinins and possessing the *A* antigen in the serum, is dominant over its absence (*an*), in which the antibodies rather than the antigen are present in the serum. Matings of alpha animals (*anan*) always breed true. The present data bring the total of individuals examined from this type of mating to 245.

Large numbers of backcross progeny have been obtained as a part of experiments designed to determine the chromosomal relations of this gene. These will be reported later. Twenty-three of the 261 alpha animals obtained were of the irregular type as differentiated by the "inhibition" test. This is almost exactly the same proportion previously obtained from this mating. When these 23 animals are reclassified under the *An* phenotype, the departure from equality of the two classes is not significant (Mating 2).

Similarly among 61 additional F_2 animals, eight of the 27 alpha animals (29.6 per cent) are irregular. When corrected for this aberration the ratio shown in mating 3 is that of the typical monohybrid F_2.

As to the possible genetic nature of the irregular alpha character the evidence is as yet incomplete. Two facts are highly suggestive. First, all irregular alpha individuals thus far obtained trace their origin to one or the other of two families, either the *A* race of Castle or to family III, an inbred New Zealand White. Second, in the backcross matings (*Anan* × *anan*) a significantly greater proportion of the progeny derived from the particular heterozygous parents (*Anan*) which possessed the irregular antibodies also showed irregular antibodies, as compared with progeny of heterozygous parents which were not irregular alphas. More critical tests of the inheritance of this serological modifying factor are now in progress.

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TABLE I. Showing the matings involving alpha (*an*) and non-alpha or the *A* character (*An*).

Mating	Phenotypes *An*	Phenotypes *an*	Total	No. & % irreg.'s	X^{2*}	P
1. *anan* × *anan*	—	95	95	—		
2. *Anan* × *anan*	250	238	488	23 (*8.81*)	.295	.50
3. *Anan* × *Anan*	42	19	61	8 (*29.6*)	1.23	.30

*After correction for irregulars.

COLCHICINE INDUCED TETRAPLOIDY

in *Delphinium Cardinale*

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University of California, Los Angeles, California

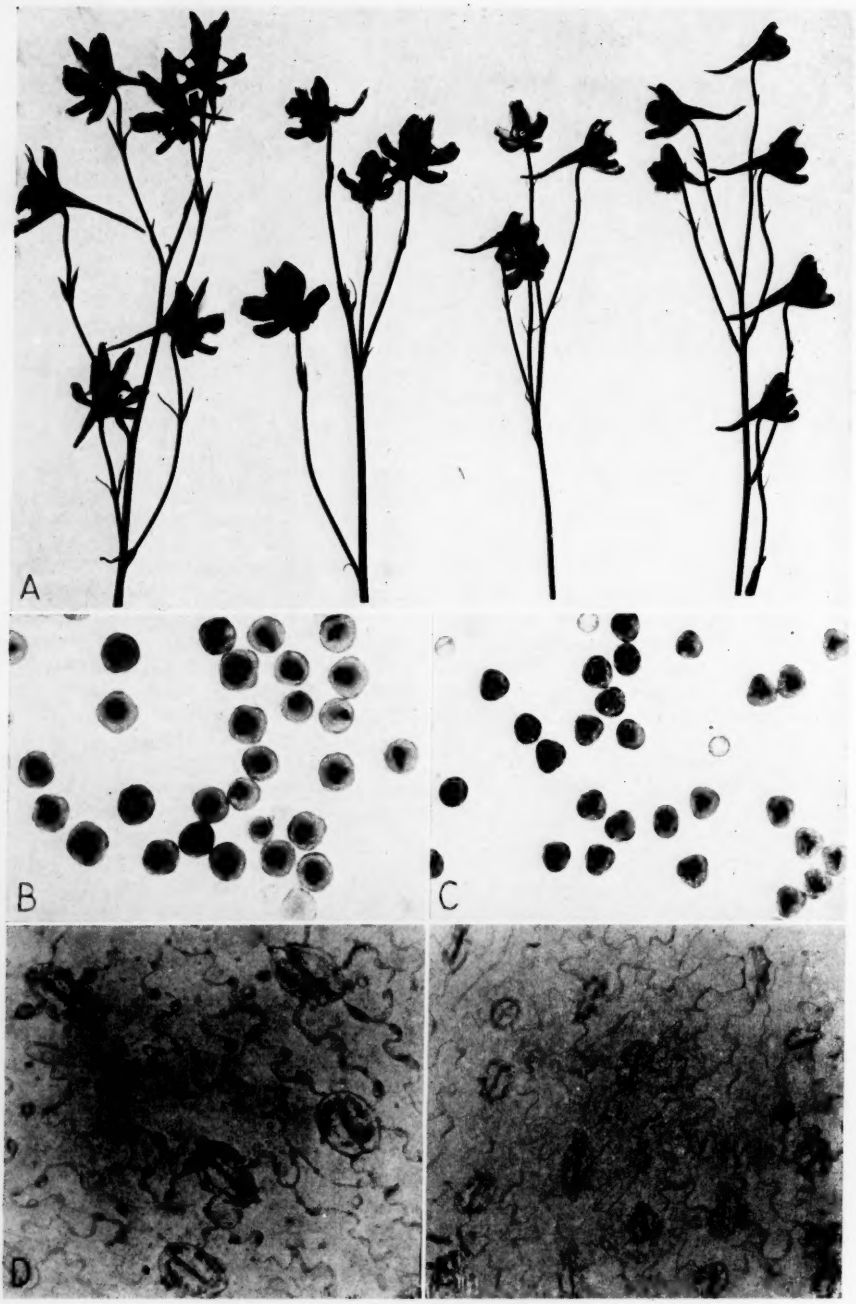
DELPHINIUM cardinale, Hook. is a perennial, endemic to cismontane southern California and Lower California. It is diploid ($n = 8$). Like most delphiniums of the Southwest, the plant becomes completely dormant after the seed matures and remains so until the following winter, when growth again begins soon after the first rains. Not only has it been shown to be highly resistant to powdery mildew, (*Erysiphe polygoni*[3]) but its flowers are of a color not yet present in the perennial cultivated delphiniums, being a cardinal red which occasionally varies through salmon and orange to yellow. The transfer of these features to the cultivated delphiniums by means of hybridization, therefore, seemed desirable.

Cytologically the perennial cultivated delphiniums fall in three groups, namely: the diploid group, chiefly represented by derivatives of *D. grandiflorum* L. ($n = 8$); the tetraploid group, probably largely derived from the *D. elatum* L. group of species ($n = 16$); and the hexaploid group, represented by the "Belladona" and "Lamartinii" varieties ($n = 24$) which are apparently allo-hexaploid hybrids between the two first named groups.[2]

As the tetraploid delphiniums, under various trade and horticultural names, are by far the most commonly cultivated and most important commercially, it seemed desirable to begin hybridization with this group.

A cross between diploid *D. cardinale* and a member of the tetraploid group might conceivably yield only triploid hybrids which would most likely be sterile. On the other hand, if a tetraploid *D. cardinale* were available, one should expect tetraploid hybrids from such a cross which might possibly be fertile. The results obtained with *D Ruysii*[2] support such an assumption. Therefore, to facilitate the transfer of desirable genes from *D. cardinale* to the tetraploid garden delphiniums it was decided to attempt the doubling of the chromosome number of *D. cardinale* by means of colchicine.

In the spring of 1938 several hundred seeds of *D. cardinale* were treated with colchicine. The concentrations used ranged from 0.1 per cent to 1.0 per cent and the length of treatment from two to 48 hours. The seeds were planted in soil in the greenhouse immediately after the treatment. Germination of the treated seed was rather poor, only about 100 plants were obtained, all of which were transferred to the field as soon as they were large enough. Most of the seedlings showed typical colchicine effects[1] in the cotyledon stage but as the true leaves appeared these abnormalities gradually became less severe. The majority of the plants did not flower until the summer of 1939. They all appeared to be normal, and examination of the size of pollen grains and stomata failed to disclose any differences from those of the untreated group. As *D. cardinale* often is slow in germinating, sometimes not germinating until the second year, all the seed pots were left in the lath house after the first lot of seedlings was removed. About six months later an additional seedling appeared. This seedling not only showed the typical colchicine effect in the cotyledon stage but the true leaves also were much thicker and somewhat twisted as compared with normal plants. It grew very slowly and failed to bloom in 1939. Chromosome determinations made on root tips from this plant revealed that it had diploid roots but the stomata were somewhat larger than normal. In 1940 two weak



DIPLOID AND TETRAPLOID COMPARED

Figure 18

A—Tetraploid flowers on the left, diploid flowers on the right. (One-half natural size.) *B*—Pollen from the tetraploid. *C*—Pollen from diploid. *D*—Stomata from tetraploid. *E*—Stomata from diploid. *B-E* all × 200.

shoots appeared, still showing some colchicine effect but not as much as in the previous year. The shoots were removed and an attempt was made to root them. One rooted and when chromosome determinations were made on its root tips the somatic number was found to be 32 instead of 16 as in the previous year. The plant reached a height of only six inches instead of the normal three to six feet and produced only three flowers. However, they were somewhat larger than normal. The pollen grains were of about twice the volume of those from normal diploid plants. In 1941 this plant attained a height of six feet but unfortunately the stem was broken during a rain storm, so that no meiotic studies could be made. Counts made on root tips proved the plant was still tetraploid. In 1942 it reached a height of four feet. The flowers and pollen were somewhat larger than those of untreated plants. Examination of meiosis confirmed that the plant was tetraploid (Figure 19*C, D*).

In the autumn of 1940 three hundred seeds were treated with colchicine under the same conditions as described above. Germination of the treated seed was again very poor, and all the seedlings that did survive gradually became normal. The untreated seed germinated well, so the differences in germination were obvioulv due to the colchicine treatment. Two different types of treatment were then tried.

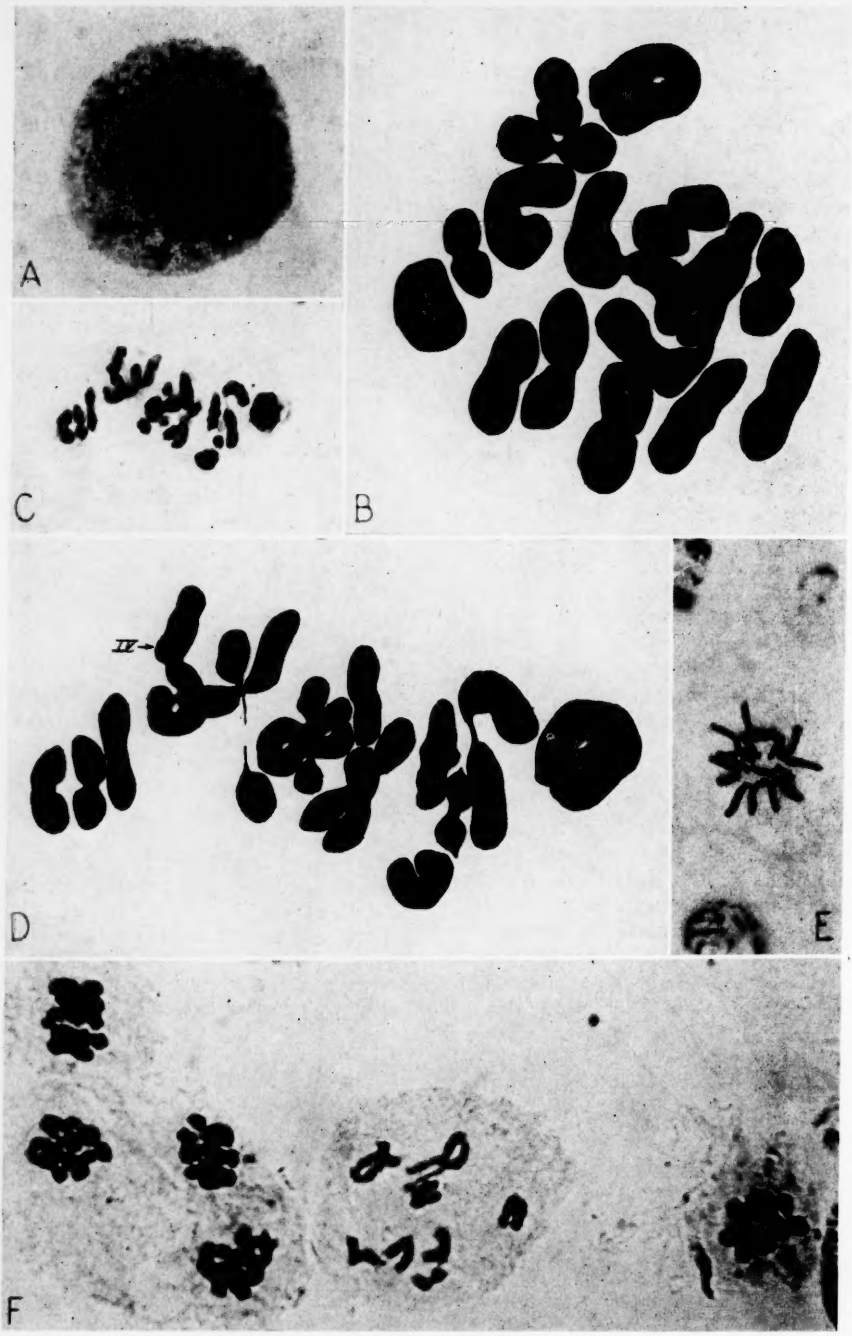
Seedlings of *D. cardinale* which had been germinated the year previous and started into growth again after a summer's dormancy were freed from soil and placed in different concentrations of an aqueous solution of colchicine so that only the larger leaves remained above the surface. Length of immersion varied from a half hour to eight hours. After treatment they were again planted in soil. The treatment proved ineffectual for, although most of the plants showed some effect of the treatment, such as stunted and twisted growth and numerous tetraploid roots, they all eventually became normal.

Treatment of seedlings still in the cotyledon stage was then tried. In November, 1940, four pots were planted with 50 seeds each. After germination the seedlings in three pots were treated while those in the fourth were retained as a control. When the cotyledons were fairly well matured and the plumule barely visible, a drop of 0.1 per cent aqueous solution of colchicine was placed directly on the plumule by means of an eye dropper. The following day the seedlings in two pots were given an additional drop of colchicine and on the third day the seedlings in one of these were given a third drop. The numbers of seedlings thus treated were 33, 33, and 34 respectively. All seedlings that appeared after treatment were later added to the control group.

The treated seedlings developed greatly swollen hypocotyls in the next few days. When the true leaves appeared they were thickened and contorted. Compared with the untreated seedlings the growth rate was greatly reduced. At the end of June the seedlings were allowed to dry out and become dormant.

The following November the seedlings were again started into growth. As the leaves appeared it was evident that only a few seedlings still showed the effect of the treatment. In fact only fifteen, five from each treatment, looked promising enough to be retained in the greenhouse along with five control plants whereas the others were planted in the field.

As the plants in the greenhouse approached full size all but one appeared normal in growth and habit. This plant had very thick twisted leaves and was greatly reduced in size. As soon as buds were available on these plants the chromosome number was determined. The determinations were made from temporary aceto-carmine smears of pollen mother cells prepared in the usual way. Of the fifteen treated plants eight were tetraploid and seven diploid. The five control plants were all diploid. On the other hand, somatic determinations



DETAILS OF MEIOSIS

Figure 19

A—Photomicrograph of first metaphase in dividing pollen mother cell (PMC) from tetraploid showing 16 bivalents, × 900. *B*—Camera Lucida drawing of *A*, × 2400. *C*—Photomicrograph of first metaphase in dividing PMC from tetraploid showing one tetravalent and 14 bivalents, × 900. *D*—Camera Lucida drawing of *C*, × 2400. (The tetravalent is marked by IV.) *E*—Somatic division in root tip cell from diploid plant showing 16 chromosomes, × 900. *F*—Diakinesis and metaphase in dividing pollen mother cells from diploid plant showing eight bivalents, × 900.

made on root tip material showed that all fifteen plants had diploid roots. All somatic counts were made from permanent preparations stained by gentian violet and safranin according to Stockwell's modification of Fleming's triple stain schedule.[4] Examination of pollen and stomata revealed the size differences shown in Figure 18*B-E*. Pollen examinations were likewise made on all the plants planted in the field. Only one plant had the larger pollen of the tetraploids in the greenhouse. Pollen mother cell smears from this plant showed that it too was tetraploid. The proportion of tetraploids obtained in relation to the number of seedlings treated was 3/33, 2/33 and 4/34 for the one drop, two drop and three drop treatments respectively.

The average overall size of the tetraploid plants was somewhat greater than that of the diploids and they were about two weeks later in flowering. Although the leaves were somewhat thicker and more leathery, they could not be depended upon as a means to distinguish the tetraploids from a group of undetermined plants among which tetraploids might be expected.

The flowers generally were larger on the tetraploids than on the diploids (Figure 18*A*) but, as flower size varies to some extent with the position of the flower on the plant as well as with the age and general condition of the plant itself, especially with respect to the nutrient level, flower size does not seem to be a good indicator of tetraploidy.

On the other hand, stomata and pollen size appear to be dependable characteristics on which to base at least a preliminary separation.

The chromosomal complement in *D. cardinale* consists of two long chromosomes with sub-median centromeres, four medium size with sub-terminal centromeres and two short chromosomes with almost terminal centromeres, (Figure 19*E*).

Although no exhaustive study of meiosis in these tetraploids has as yet been possible, the most striking feature apparent in the preparations examined thus far is the almost complete absence of multivalents. In 107 cells that were sufficiently well spread out to permit complete counts 4 contained two tetravalents and 12 bivalents, 23 contained 1 tetravalent and 14 bivalents (Figure 19 *C, D*), and 80 cells contained bivalents only. (Figure 19*A, B*).

In late diplotene and diakinesis only one chiasma per bivalent could be seen with certainty for the short and medium size chromosomes. (Figure 19*F*). In only one cell from an untreated diploid plant two of the medium sized bivalents appeared as though they might have had two chiasmata each but this could not be verified. In the case of the long chromosomes, on the other hand, two chiasmata per bivalent was the rule and occasionally three or possibly four. This was true in both diploid and tetraploid plants.

There is of course the possibility that the very cells in which all units could not be counted actually owed their more or less clumped appearance to the presence of a higher number of multivalents but the appearances of the diplotene and diakinesis stages do not support this supposition. As all the observed tetravalents involved the long chromosomes it seems more likely that the low frequency of tetravalents is due to the low chiasma frequency.

Seed production on selfing the tetraploids was approximately 60 per cent of that produced by the corresponding diploids. The seeds from the tetraploid plants were considerably larger than the seeds from the diploid plants, but the difference was not so striking as in the case of the pollen grains and stomata. Crosses made between diploids and tetraploids resulted in a few apparently good seeds when the tetraploids were used as seed parents. From the reciprocal cross very few seeds of doubtful viability were obtained.

Crosses between tetraploid *D. cardinale* and several members of the tetraploid group of the cultivated delphiniums have resulted in a fair amount of seed but as yet only three hybrids have been

obtained. The results from planting more of these hybrid seeds, as well as the behavior of the hybrids already obtained, will be reported later.

We are indebted to Mr. C. A. Schroeder for help in the preparation of the photographs.

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✦

RACE DIFFERENCES IN INTELLIGENCE

(*Concluded from page* 174)

ter, for example, summarizes the literature in the following trenchant fashion:

". . . all results show the Negro decidedly inferior to the white on standard intelligence tests. This difference is present among infants, elementary school children, high school pupils, university students and adult men. It occurs on Binet tests, on verbal group tests, on non-verbal group tests, and on performance tests. These results are sufficiently numerous and consistent to point to a racial difference in intelligence. The overlapping of the two races is great, and the most liberal estimate seems to be that at most 25 per cent of the colored reach or exceed the median intelligence of the whites. No qualitative difference in intelligence between the two races can explain this marked quantitative difference."

It is an obvious extension of the preceding results to conclude that inferior achievement with respect to intelligence tests constitutes inferiority in intelligence itself. This conclusion need not follow, however, unless it can be shown that the tests actually measure native intelligence, at least within reasonable limits. This is denied by many investigators, especially by cultural anthropologists. As applied to Negroes, there are undoubtedly factors in the tests that operate to their disadvantage. This results primarily from the fact that intelligence scales have been standardized almost entirely upon white populations. Since the cultural and other social factors constituting the environment of Negro children are decidedly inferior to those surrounding white children, it must be considered unreasonable to expect them to do as well as white children on scales that were prepared on the basis of the much richer experience of white children. That there is some force to this argument appears clear from the results of Klineberg's studies with groups of migrant Negro children in New York City. Classifying these children —all from the South—in groups corresponding to the length of residence in New York City, he showed that the average intelligence of the groups rose as the period of residence in New York City increased, and came closer and closer to the averages of white children in the same schools. He therefore concluded that it is unlikely that there are any real quantitative differences in intelligence between Negro and white children.

It appears to me that Klineberg's opinion, which may turn out to be correct, nevertheless goes beyond the facts of the case. It is clearly a possibility that the trend noted by Klineberg will finally level off and approach some asymptotic value. The crucial question therefore is —where will the asymptote appear in relation to that for whites? It may well be, that after a rise due to better adjustment in general, the average degree of intelligence of Negro children will become constant at a value below that of the white children. Certainly, Klineberg does not go so far as to suggest that the level of intelligence of the Negro children will ultimately exceed that of the white children. The burden of proof would seem to rest upon him, and though he and others have shown that corrections must be made for the adverse effects of poor environment upon the development of general intelligence, it has not been shown that such corrections will eliminate all racial differences in such respect.

Other racial groups that measure low on intelligence scales are American Indians and Mexicans. On the other hand, Chinese and Japanese rank almost as high as whites. It can hardly be maintained that general environmental conditions favored these two groups. Consequently, we must admit a weakening of the value of the argument with respect to the influence of environment on the test results. The same conclusion follows when we consider the results of intelligence tests of white children. Certain groups, especially Italians and Poles, have done very poorly. On the other hand, Jewish children have ranked as high or higher than native children of native parentage.

On the whole, then intelligence tests have shown that there are large group differences in intelligence, and that certain races, and certain nationalities, rank high on such scales, and others rank low. I leave open the question whether from such results we are justified in concluding that the frequency of mental defect varies in the same manner. This represents not undue caution, but the important fact that mental defect cannot be measured solely on the basis of the intelligence quotient.

* * *—Benjamin Malzberg, Ph.D., Department of Mental Hygiene, Albany, New York. *Am. Jour. Ment. Deficiency*, January, 1943.

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